UNITED STATES DISTRICT COURT
FOR THE EASTERN DISTRICT OF PENNSYLVANIA

IN RE: NATIONAL FOOTBALL LEAGUE PLAYERS' CONCUSSION INJURY LITIGATION	No. 2:12-md-02323-AB MDL No. 2323
Kevin Turner and Shawn Wooden, *on behalf of themselves and others similarly situated,* Plaintiffs, v. National Football League and NFL Properties LLC, successor-in-interest to NFL Properties, Inc., Defendants.	CIVIL ACTION NO: 14-cv-0029 **Hon. Anita B. Brody**
THIS DOCUMENT RELATES TO: ALL ACTIONS	**DECLARATION OF JULIE ANN SCHNEIDER, M.D.**

I, JULIE ANN SCHNEIDER, M.D., hereby declare as follows:

1. If called as a witness, I could and would testify competently to the facts herein.

I. Qualifications

2. I am a Professor of Pathology (Neuropathology) and of Neurological Sciences at Rush University Medical Center in Chicago, Illinois. In addition to my teaching responsibilities, I also am a researcher and clinical neuropathologist and neurologist who diagnoses neurodegenerative diseases and other neuropathologies. My complete *curriculum vitae* is attached as Exhibit A. I highlight

here some of my qualifications, experience, and research relevant to the opinions I express below.

3. I obtained a Bachelor of Science in Biology-Psychology from the University of Illinois in 1983, *magna cum laude*. I obtained a Doctor of Medicine with Honors from the University of Illinois, School of Medicine in 1987. I completed my residency training in Neurology at the University of Chicago in 1991. I completed a fellowship in Neuropathology at the University of Chicago and at Emory University in Atlanta, Georgia in 1993, where I focused on neurodegenerative diseases. I also received a Master of Science in Clinical Research with a special emphasis in Epidemiology from Rush University Medical Center in 2003.

4. I am licensed by the National Board of Medical Examiners and am board certified in both Neuropathology and Neurology.

5. I have been on the faculty at Rush University Medical Center since 1996. Currently, I train pathology residents to diagnose neurodegenerative and other complex pathologic diseases in dementia and movement disorders by examining patients that come to the autopsy service at Rush University Medical Center. I also teach neuropathology to pathology, neurology, and neurosurgical medical residents.

6. In addition to my responsibilities as a university professor, as a clinical neuropathologist, I regularly examine brains of deceased patients with complex neurodegenerative diseases and the brains of deceased participants of longitudinal studies on aging and dementia. I also pathologically diagnose both common and rare neurodegenerative diseases related to cognitive and motor impairments. As a board certified neurologist I also see patients with memory complaints and neurobehavioral disorders at the Rush Memory Clinic, where I have been an attending physician for the past 18 years.

7. I serve as Associate Director, Neuropathology Core Leader, and Senior Neuropathologist of the Rush Alzheimer's Disease Center, one of 29 Alzheimer's Disease Centers in the nation funded by the National Institutes of Health ("NIH").

8. I currently am part of 17 investigative teams with grants from various organizations, including five projects being funded by NIH. I am the principal investigator in an epidemiologic study of TDP-43 in aging and dementia designed to determine whether age-related TDP-43 pathology represents a separate pathologic process associated with a dementia syndrome with a distinct cognitive phenotype and specific genetic risk factors that are separate from Alzheimer's disease. TDP-43 is a protein that deposits in frontotemporal lobar degeneration, amyotrophic lateral sclerosis ("ALS"), and Alzheimer's disease and aging, and that also has been associated with chronic traumatic encephalopathy ("CTE"). I also am part of investigative teams studying the clinical profile of Parkinson's disease pathology (Lewy body pathology), the optimization of neuropathological assessment of Alzheimer's disease, and the risk factors, pathology and clinical expressions of Alzheimer's disease.

9. I am an invited member of the expert panel that convened to provide new guidelines for the criteria for a pathologic diagnosis of Alzheimer's disease in 2012. B.T. Hyman, et al., *National Institute on Aging-Alzheimer's Association Guidelines for the Neuropathologic Assessment of Alzheimer's Disease*, 8 Alzheimers Dement. 1 (2012).

10. I am on the editorial boards of three scientific journals, including the Journal of Neuropathology and Experimental Neurology. I peer-review manuscripts for over 25 scientific journals, including Neurology, Lancet, the Journal of the America Medical Association ("JAMA"), and Annals of Neurology.

11. I serve on the external advisory boards of five organizations, including the University of California San Francisco Neurodegenerative Brain Bank, the

New York University Alzheimer's Disease Core Center, and the Boston University Alzheimer's Disease Core Center.

12. I am an author of over 180 peer-reviewed scientific articles. Recently, Thomson Reuters recognized me for being among the top 1% of highly cited researchers in the world in the area of neuroscience and behavior.

13. In my role as a consulting expert for the NFL Parties in this litigation, I am being compensated for my time at my standard hourly rate.

II. Assignment and Summary of Opinions

14. I have reviewed the Settlement Agreement and Exhibits 1, 2, and 3 to the Settlement Agreement—the Injury Definitions, the Test Battery and Specific Impairment Criteria, and the Monetary Award Grid. I also have reviewed certain objections to the proposed Settlement relating to medical or scientific issues, including the supporting declarations of Dr. Robert Stern, Dr. Samuel Gandy, and Drs. Brent Masel and Gregory O'Shanick.

15. I have been asked to provide medical and scientific testimony regarding the challenges of proving a causal link between concussions or subconcussive hits, i.e., mild repetitive traumatic brain injury ("TBI"), and CTE. I also have been asked to provide medical and scientific testimony relating to some of the objections regarding CTE.

16. As discussed in detail below, it is my opinion, based on the state of the science regarding the clinical pathologic syndrome known as CTE, that there are very significant gaps in the understanding of the possible association between mild repetitive TBI and the clinical syndrome and brain pathology reported as CTE. In addition, there are significant gaps in our understanding of the presumed risk factors for CTE, and the diagnostic and clinical profile of presumed CTE. Therefore, from a scientific standpoint, I think it would be difficult for plaintiffs to establish a causative relationship between their mild repetitive TBI during NFL play and the clinical and pathologic changes

reported to be associated with CTE. Similarly, even more difficult would be for plaintiffs to establish from a scientific standpoint that mood and behavioral symptoms that they may be experiencing are being caused by CTE. Finally, it is my opinion that, putting aside issues of causation, if the Settlement provided compensation for mood and behavior changes, a large number of players who are not truly manifesting symptoms of CTE while living would be compensated. Indeed, this is also true for cognitive symptoms currently reported to be associated with CTE, given that dementia, Alzheimer's disease, and other comorbid conditions are extraordinarily common in aging in the absence of CTE.

17. All of the opinions expressed herein are offered to a reasonable degree of medical certainty and are consistent with opinions that I would offer in my clinical practice.

III. Opinions

A. The Scientific Community's Understanding of the Association Between TBI and CTE

18. I have reviewed the objections regarding CTE, including the medical declarations in support of those objections. I understand that certain objectors criticize the fact that brain changes of CTE are not compensated under the settlement after July 7, 2014. I also understand that if these cases proceeded through litigation, the retired NFL players would be required to prove that the concussions and subconcussive hits, *i.e.*, mild repetitive TBI, that they experienced while playing football in the NFL caused them to develop the brain changes and impairments reported to be associated with CTE. As described in more detail below, it is my scientific opinion that the retired players would face difficulties in establishing a causal relationship between mild repetitive TBI and the brain changes of CTE. This is largely because the scientific community's understanding of CTE is preliminary and, in my opinion, we do not yet understand the association between mild repetitive TBI and CTE. Specifically, we as a

scientific community do not know whether athletes, both with and without TBI, and both with and without cognitive impairment, may harbor these brain changes. Thus, the scientific community has not established a specific association between mild repetitive TBI and the brain changes of CTE.

19. In describing the challenges that I believe the retired players would have in proving that mild repetitive TBI causes the brain changes of CTE, I will first explain the state of the science with respect to CTE. I will then contrast the state of the science regarding CTE with the state of the science regarding Alzheimer's disease, which I believe is a useful comparison for understanding the challenges faced by retired players in establishing causation. Finally, I will explain why, in my scientific opinion, I believe that the Settlement compensates the key symptoms currently reported to be associated with CTE to date.

i. The State of the Science Regarding CTE

20. The state of science regarding repetitive head trauma and CTE is incomplete. This applies both on a neuropathological level, in terms of understanding how repetitive head trauma and CTE impacts the brain, and on a clinical level, in terms of understanding the risk factors for CTE, its clinical expression (what symptoms the disease manifests), and how to establish a diagnosis of CTE in living individuals based on a specific clinical profile. Because the research in these areas is incomplete, I believe that we as a scientific community cannot yet make definite conclusions as to the impact of CTE on the brain, the risk factors of CTE, or the clinical expression of CTE.

21. From a neuropathological perspective, as of today, CTE can only be diagnosed post-mortem. This simply means that a neuropathologist must physically look at an individual's brain post-mortem (after death), determine if something called the "tau protein" (abnormally phosphorylated tau protein) is present, and assess whether the tau protein is present in a reportedly unique distribution pattern. It is important to note that abnormal tau protein is also a primary component of other neurodegenerative

diseases, most notably Alzheimer's disease and frontotemporal lobar degeneration, as well as the fact that these neurodegenerative diseases have been reported in conjunction with CTE.

22. CTE is reportedly characterized by distinct neuropathological findings. *See* Baugh, C., *et al.*, *Chronic Traumatic Encephalopathy: Neurodegeneration Following Repetitive Concussive and Subconcussive Brain Trauma*, 6 BRAIN IMAGING & BEHAVIOR 244 (2012). The most important of these findings is that CTE is neuropathologically characterized by aggregation and accumulation of variations of a protein known as tau, specifically hyperphosphorylated tau. *See id.*

23. However, aside from the existence, prevalence, and pattern of the abnormal tau protein accumulation, there are still many uncertainties as to the neuropathology of CTE. To date, there are very few studies that have attempted to create pathological classifications of CTE. These studies have reached similar conclusions regarding neuropathological findings, but have also found some significant differences, including the hallmark features of CTE, the location of the accumulation of tau in the brain, and the neuropathological impact of the alleged progression of CTE. *See* Gardner, *et al.*, *Chronic Traumatic Encephalopathy in Sport: A systematic Review*, 48 Br. J. Sports Med. 84 (2014) (the "Gardner Study") (comparing the findings of doctors from Boston University, including Dr. McKee and Dr. Stern, with findings of Dr. Omalu).

24. In addition to not yet fully understanding the neuropathology of CTE, many other questions regarding CTE remain unanswered, including the risk factors for CTE, the relationship between TBI and CTE, and the diagnostic and clinical profile of CTE. These questions remain because clinical studies regarding CTE, though evolving, to date have been incomplete. In fact, there have only been a handful of clinical studies relating to CTE. These studies are extremely important in guiding future research, but there are two main issues with these studies. The first is that, put simply, there are just too few studies using state of the art scientific and medical methods to make sound

conclusions. The second limitation is that the studies—because they are case reports—are inherently limited and prone to bias. I will address each limitation in turn.

25. First, the scientific community agrees that in terms of sheer numbers, there have not yet been enough well conducted studies with large enough numbers to understand CTE with scientific certainty. *See, e.g.*, Report on the Neuropathology of Chronic Traumatic Encephalopathy Workshop, National Institutes of Health, (Dec. 5-6, 2012), *available at* http://www.ninds.nih.gov/news_and_events/proceedings/201212_CTE_workshop_report.htm ("[V]ery few studies have looked at biomarkers that address the long-term disease processes of CTE. Much work remains to identify useful, validated biomarkers that provide information about the injury mechanisms of CTE."). CTE has only been studied on fewer than 200 brains in a very limited number of retrospective studies in the past ten years. Moreover, sample sizes in individual studies are too small and methodologies too inadequate for the scientific community to make key conclusions regarding CTE and prospective studies are needed.

26. Second, the studies associated with CTE, to date, are inherently limited and likely to suffer from multiple types of bias that are inherent to retrospective studies and studies without controls (*e.g.*, recall bias and selection bias). There are no published double-blind randomized control trials, prospective studies, cross-sectional studies, or even case control studies regarding CTE. The handful of studies that have been conducted relating to CTE are case reports or case series. These studies are important for generating scientific hypotheses to be tested in long-term, prospective studies. However, it is important to understand that the scientific community does not typically make medical or scientific conclusions based on case series or case reports alone. Some of the typical limitations associated with case series include selection bias, a lack of a proper control group, retrospective collection of data, and information bias.

27. Because of the limited number of studies available, and the nature of the case reports that have been published, it is my opinion that we do not know enough about CTE to adequately understand its risk factors, the relation between repetitive TBI and CTE, or the diagnostic and clinical profile of CTE. Thus, one should be cautious making assumptions about a causal association between mild repetitive TBI, *i.e.*, concussions or subconcussive brain injuries, and CTE until well-designed scientific studies are conducted. Similarly, one should be cautious making assumptions regarding symptoms that constitute the diagnostic and clinical profile of CTE until well-designed scientific studies are conducted.

28. My opinions are shared by the scientific community, *i.e.*, that "[o]wing to the nature of the case reports and pathological case series that have been published it is not possible to determine the causality or risk factors [of CTE] with any certainty." P. McCrory, et al., *Consensus statement on concussion in sport: the 4th International Conference on Concussion in Sport held in Zurich*, 250 Br. J. Sports Med. 58 (2013) (the "McCrory Study") ("[T]he speculation that repeated concussion or subconcussive impacts cause CTE remains unproven. The extent to which age-related changes, psychiatric or mental health illness, alcohol/drug use or co-existing medical or dementing illnesses contribute to this process is largely unaccounted for in the published literature."); *see also Sports Related Concussions in Youth: Improving the Science, Changing the Culture, Institute of Medicine*, (Oct. 30, 2013), *available at* http://www.iom.edu/Reports/2013/Sports-Related-Concussions-in-Youth-Improving-the-Science-Changing-the-Culture/Report Brief103013.aspx ("[I]t remains unclear whether repetitive head impacts and multiple concussions sustained in youth lead to long-term neurodegenerative diseases, such as chronic traumatic encephalopathy.").

ii. The State of the Science Regarding The Alleged Symptoms of CTE

29. I have reviewed the objections regarding CTE, and I understand that certain objectors argue that mood and behavioral symptoms are part of the diagnostic and clinical profile of CTE and, therefore, should be compensated under the Settlement Agreement. As discussed above, it is my opinion that assumptions regarding symptoms that constitute the diagnostic and clinical profile of CTE are premature. Thus, it is premature to consider mood and behavioral symptoms as part of the diagnostic and clinical profile of CTE.

30. I have reviewed the declaration of Dr. Robert Stern, the primary expert who filed a declaration in support of the objections. In his declaration, Dr. Stern describes the alleged clinical and diagnostic profile of CTE. Dr. Stern's research, which is conducted with other doctors at Boston University, including Dr. Ann McKee, constitutes important research in the field of CTE at this time. However, the studies that he has conducted with his colleagues are case reports that suffer from the kinds of limitations described above. I will provide a brief discussion of the limitations of those studies as well.

31. The first study, the McKee Study, *see* Ann McKee, Robert Stern, et al., *The Spectrum of Disease in Chronic Traumatic Encephalopathy*, 136 BRAIN 43 (2013) ("McKee Study"), included an analysis of the brains of 85 subjects, including former athletes, military veterans and civilians with a history of repetitive mild TBI. *See* McKee Study at 55. Eighteen gender-matched individuals without a history of repetitive mild TBI served as the control group. *See id.* The study found evidence of CTE in 68 subjects' brains. Symptoms relating to these subjects were recounted from the subjects' family (this was conducted as to only approximately half of the 85 individuals; the other approximately half of the subjects were excluded from consideration, possibly because the researchers were unable to speak to the subjects' next of kin). A medical record

review of each of the 45 subjects was also conducted. *Id.* The study broke CTE into four progressive stages. Based on interviews of the family members, the study attempted to assess what symptoms were associated with each stage of CTE. Ultimately, the study concluded that although CTE Stages III and IV progress to dementia, *see id.* at 55-63, CTE I and II were associated with mood and behavioral symptoms and some cognitive impairments. The study did find that many of the subjects that had CTE also had co-morbid disease, which includes Alzheimer's, Parkinson's, ALS and frontotemporal dementia.

32. The second study, the Stern Study, *see* Robert Stern, et al., *Clinical Presentation of Chronic Traumatic Encephalopathy*, 81 NEUROLOGY 1122 (2013) (the "Stern Study"), used the same methodology as the McKee Study to assess the clinical symptoms allegedly associated with individuals diagnosed post-mortem with CTE. The Stern Study also relied largely on the same subjects as the McKee Study for the diagnosis of CTE. The brains of 81 subjects were examined post-mortem for neuropathology consistent with CTE. The authors then contacted the next of kin for a retrospective report of the subjects' symptoms. However, like in the McKee Study, only certain subjects' families were contacted regarding the subjects' symptoms. In addition, a number of potential subjects were excluded from this aspect of the study, including individuals with the presence of comorbid disease. Despite the fact that the study concluded that there "may be 2 different clinical presentations of CTE, with one initially exhibiting behavioral or mood changes, and the other initially exhibiting cognitive impairment," *see id.* at 1124, of the 33 symptomatic patients in the group, many, if not most, experienced some combination of memory impairment, executive dysfunction, attention and concentration difficulties, language impairment and visuospatial difficulties—which are addressed under the Settlement.

33. Both studies—the McKee Study and the Stern Study—are of limited value in allowing the scientific and medical communities to make conclusions

about the diagnostic and clinical profile of CTE (or the causes of CTE) because both studies are case series, which, as discussed above, are more useful in generating hypotheses than testing them. The limitations of the McKee and Stern Study are inherent in the methodology employed in both studies.

34. First, the sample of patients examined in each study is a limitation. In both studies, the participants were self-selected because they (or their families) donated their brains to the brain bank at Boston University to determine whether the subjects had CTE in their brains. This type of self-selection is referred to as selection bias in the scientific and medical communities. This study design severely limits the ability to draw any definitive conclusions

35. Second, the studies did not have proper control subjects to compare against the participating subjects. More specifically, the studies did not include a comparison group of non-athletes who experienced head trauma, athletes without TBI, athletes without CTE, or even athletes with TBI but without concerns regarding impairment. Understanding how the symptoms and brain changes of the self-selected subjects compare to the symptoms and brain changes of proper controls is essential to understanding the diagnostic and clinical profile of CTE.

36. Third, the process through which information was collected regarding subjects' symptoms was inherently limited. Next of kin, without any medical training in assessing symptoms, were asked to recall symptoms that occurred months or years earlier. Moreover, the family members who were interviewed likely suspected that their deceased relatives had CTE because of concerns about cognition or behavior, which creates potential bias in their recollections. This is known as information or recall bias. Information and recall bias is likely to overestimate the presence and prevalence of risk factors and cognitive/behavioral changes.

37. Fourth, the studies do not control for other confounding factors, such as genetic predisposition, sleep apnea, higher BMI, lifestyle change, obesity, age,

substance abuse, or cerebrovascular or cardiovascular disease. Until further prospective studies can examine these associations more closely, and control for these additional possible risk factors, it will be difficult for the scientific community to reach a consensus regarding the diagnostic and clinical profile of CTE.

38. Finally, the studies on their own do not answer key questions necessary to understand essential aspects of CTE, including the diagnostic and clinical profile of CTE: What is the prevalence and incidence of CTE in the general population? What is the prevalence and incidence of CTE in a randomly selected group of athletes with and without repetitive head trauma? How frequently is the pattern of abnormal tau protein considered consistent with a neuropathological diagnosis of CTE found in a patient's brain who was asymptomatic during life? How often are cognitive changes in athletes with repetitive TBI unrelated to CTE but rather related to alternative risk factors and other prevalent diseases, such as Alzheimer's disease? Until these questions, and many others, are answered, the scientific and medical communities will be unable to form conclusions about the possible causes and risk factors of CTE, the association between repetitive head injury and CTE, or the diagnostic and clinical profile of CTE.

39. Even assuming that these studies were later proven to be true (*i.e.*, that the diagnostic and clinical profile of CTE includes mood and behavioral symptoms), there is the added concern that many of the reported neurobehavioral symptoms are quite common in the general population. For example, headaches, depression, aggression, explosivity, and even cognitive impairment could have been due to any number of other, independent risk factors, including, but not limited to the risk factors referenced above, such as genetic predisposition, sleep apnea, higher BMI, lifestyle change, obesity, age, substance abuse or cerebrovascular or cardiovascular disease. Even where CTE is present, and even assuming the diagnostic and clinical profile of CTE includes mood and behavioral symptoms, any one of these independent risk factors could have caused the subjects' mood and behavioral symptoms reported in the McKee Study and the Stern

Study. Because of this, it would be difficult, if not impossible, to say that CTE caused or materially contributed to these symptoms in any individual case. As an example, and as described below in more detail below, some of the neurobehavioral symptoms that traditionally have been believed to be part of the spectrum of clinical Alzheimer's disease are now considered risk factors for cognitive decline rather than part of the disease process. This is true of depression which is very common in aging and is a risk factor for cognitive decline and AD but is not associated with the pathology (plaques and tangles) or clinical progression of the disease.

40. Overall, based on the above, it is my opinion that the scientific community does not understand the risk factors for CTE, the association between mild repetitive TBI and alleged clinical or pathologic changes associated with CTE, or the diagnostic and clinical profile of CTE. Therefore, from a scientific standpoint, I think it would be difficult for plaintiffs to establish that mild repetitive TBI during NFL play was a primary cause of clinical symptoms and brain changes reported as CTE. I also think it would be enormously difficult for plaintiffs to establish from a scientific standpoint that mood and behavioral symptoms that they may be experiencing, such as depression, headaches, and aggressiveness, are being caused by CTE, even if a clear diagnostic profile of CTE was later established.

iii. The State of Science Regarding Alzheimer's Disease

41. In my opinion, the state of the science regarding Alzheimer's disease serves as a useful contrast to the state of the science regarding CTE. Unlike CTE, Alzheimer's disease has been studied across tens of thousands of subjects for decades in numerous long-term, prospective studies with proper controls.

42. In my opinion, the scientific community's early understanding of Alzheimer's disease was similar to the scientific community's current understanding of CTE. In early studies, cases with clinical symptoms thought to be Alzheimer's disease were studied pathologically (case reports and case series). Similar to CTE, a diagnosis of

Alzheimer's disease could initially only be made post-mortem by reviewing the neuropathology of a patient's brain. In Alzheimer's disease, the relevant proteins are the beta-amyloid protein and the abnormal tau protein (the same protein used to diagnose CTE). Because the first brain examined was in a younger person, it was first thought that Alzheimer's disease was a "presenile dementia", or early onset dementia, starting before the age of 60 years. This was later found to be erroneous, and we now know that early onset Alzheimer's disease is relatively rare and much more commonly inherited as an autosomal dominant trait (familial Alzheimer's disease), compared to the very common late onset disease. It is noteworthy that Alzheimer's disease and other dementias are present in over 10% of the persons over the age of 65 years, and rises to almost 50% after the age of 85 years old. *See* Denis A. Evans, *Prevalence of Alzheimer's Disease in a Community Population of Older Persons. Higher than Previously Reported*, 262 JAMA 2551, 2551 (1989) (finding that "[o]f those over the age of 65 years, an estimated 10.3% . . . had probable" AD, and "47.2%" of those "over 85 years" had probable AD). Thus, dementia is a very common condition even in non-athletes. Over time, the scientific community's understanding of Alzheimer's disease has evolved as new, long-term prospective studies have been completed. Today, based on these long-term, prospective studies, the diagnostic and clinical profile of Alzheimer's disease is much better understood. Based on this clinical profile, clinicians can diagnose Alzheimer's disease in living patients, *i.e.*, without a post-mortem neuropathological diagnosis, with approximately 80 to 90 percent accuracy, depending on the level of expertise of the physician.

43. Even though our understanding of Alzheimer's disease has improved markedly over the past decades, it has become clear that Alzheimer's disease is just one of many pathologies that is related to cognitive impairment in aging. This is relevant to CTE in that it is likely that persons with presumed CTE may very commonly have other brain pathologies that could independently be related to cognitive impairment.

One example is cerebrovascular disease (infarcts and vessel disease). Indeed, memory loss, the *sine qua non* of Alzheimer's disease, is now known to be a common manifestation of brain vascular diseases other than Alzheimer's disease. *See, e.g.*, Minke Kooistra, et al., *Vascular Brain Lesions, Brain Atrophy, and Cognitive Decline. The Second Manifestations of ARTerial Disease—Magnetic Resonance (SMART-MR) Study*, 35 Neurobiol. Aging 35, 35 (2014) (brain vascular diseases are associated with cognitive impairment ("e.g., memory performance, executive functioning, and information processing speed"), not only "in patients with clinical manifestations of vascular disease" but also in "healthy middle-aged and older populations."); Philip B. Gorelick, et al., *Vascular Contributions to Cognitive Impairment and Dementia: A Statement for Healthcare Professionals from the American Heart Association/American Stroke Association*, 42 Stroke 2672, 2674 (2011) ("Gorelick Study") ("Although Alzheimer disease is the most commonly diagnosed cause of cognitive dysfunction among the aged, cognitive impairment caused by vascular disease, including subclinical brain injury, silent brain infarction (SBI), and clinically overt stroke are important as independent causes and contributors to cognitive dysfunction."); Zoe Arvanitakis, et al., *Microinfarct Pathology, Dementia, and Cognitive Systems*, 42 Stroke 722, 722 (2011) (noting that brain infarcts are "common in older persons," and are associated with "lower cognition, specifically perceptual speed and semantic and episodic memory"). Adding more diagnostic complexity, we now know that older persons often have multiple pathologies in their brains (mixed pathologies) contributing to memory loss and cognitive impairment, very commonly Alzheimer's disease and infarcts, and these infarcts are often too small to be visualized by routine neuroimaging (MRI). It is often difficult during life to know whether one or multiple of these pathologies is contributing to an individual's cognitive impairment.

44. Also relevant to CTE is the fact that we also now know that one-third of older persons who die without cognitive impairment have full Alzheimer's

disease pathology, undiagnosed, in their brains. *See, e.g.*, D. A. Bennett, et al., *Neuropathology of Older Persons Without Cognitive Impairment from Two Community-Based Studies*, 66 Neurology 1837, 1841 (2006) (finding that about one-third of persons without obvious signs of dementia had intermediate or high likelihood AD); Howard Jay Aizenstein, et al., *Frequent Amyloid Deposition Without Significant Cognitive Impairment Among the Elderly*, 65 Arch. Neurol. 1509 (2008) (finding that in a community-based sample of individuals from 65 to 88 years who did not show signs of AD or mild cognitive impairment, twenty-one percent showed evidence of amyloid deposition, which is consistent with AD); Reisa A. Sperling, et al., *Toward Defining the Preclinical Stages of Alzheimer's Disease: Recommendations from the National Institute on Aging-Alzheimer's Association Workgroups on Diagnostic Guidelines for Alzheimer's Disease*, 7 Alzheimers Dement. 280, 285 (2011) (noting that ongoing studies have provided preliminary evidence "that biomarker abnormalities consistent with AD pathophysiological process are detectable before the emergence of overt clinical symptomatology and are predictive of subsequent cognitive decline."). It is believed that these individuals have cognitive reserve and are able to compensate for pathology without showing clinical symptoms. This is also relevant to CTE. It is currently not known whether some athletes in specific sports will have the reported brain changes of CTE, but not exhibit symptoms during life. Given my experience with Alzheimer's disease and other brain diseases, in my opinion it is quite likely that there can be the reported pathology of CTE without symptoms.

45. It is noteworthy that our understanding of the diagnostic and clinical profile of Alzheimer's disease continues to evolve and aspects of the profile are not easily understood by the scientific and medical communities. One example, which I believe is relevant to the complaints raised by the objectors, involves mood symptoms. For many years, the scientific and medical communities believed that changes in mood, specifically depression, were part of the diagnostic and clinical profile of Alzheimer's

disease. Today that belief has been refuted and the scientific and medical communities now understand that depressive symptoms are not part of the diagnostic and clinical profile of Alzheimer's disease. *See, e.g.*, R. S. Wilson, et al., *Change in Depressive Symptoms During the Prodromal Phase of Alzheimer Disease*, 65 Arch. Gen. Psychiatry 439, 439-45 (2008) (showing no systematic change in depressive symptoms during the prodromal or early stages of AD); R.S. Wilson, G.M. Hoganson, et al., *Temporal Course of Depressive Symptoms During the Development of Alzheimer Disease*, 75 Neurology 21, 21-26 (2010) (showing barely perceptible depressive symptoms presymptomatic and in symptomatic AD); R.S. Wilson, A. W. Capuano, et al., *Clinical-Pathologic Study of Depressive Symptoms and Cognitive Decline in Old Age*, 83 Neurology 702, 702-09 (2014) (finding that none of the neuropathologic markers of dementia was related to the level of depressive symptoms or change in symptoms).

46. I also note that there is still much to be learned about Alzheimer's disease, including the cause or causes of Alzheimer's disease, which are still not well-understood by the scientific community. In fact, despite how much is known about Alzheimer's disease today, NIH alone funds over $500 million per year in Alzheimer's-related research. *See* Estimates of Funding for Various Research, Condition, and Disease Categories (RCDC), U.S. Dept. of Health & Human Services (Mar. 7, 2014), *available at* http://report.nih.gov/categorical_spending.aspx. This is true despite the many years of extensive research into the disease.

47. Finally, I have reviewed certain objections that emphasize that the scientific community will soon be able to diagnose CTE in living people because a compound—known as a biomarker—soon will be identified that will allow for the identification of the tau protein in living people who take a PET scan. This may be true, but based on Alzheimer's disease research, the presence of a biomarker for a protein does not currently tell us whether an individual is exhibiting symptoms or the likelihood that he will experience symptoms. As noted above, neuropathologists, including myself,

regularly find amyloid protein consistent with Alzheimer's disease in the brains of patients who are asymptomatic during their lives and who never experience the symptoms of Alzheimer's disease. Moreover, the abnormal tau protein is present in multiple neurodegenerative diseases and in aging which may make interpretation of biomarker studies difficult. Finally, the tau biomarker has not been FDA approved and remains under investigation.

B. The Settlement Compensates the Key Alleged Symptoms of CTE

48. Thus far, I have explained why I do not believe that the scientific and medical communities understand the cause or causes of CTE or the diagnostic and clinical profile of CTE (*i.e.*, CTE's associated symptoms). However, even if one assumes that there is a causal relationship between mild repetitive TBI and CTE—as alleged by several objectors—in my opinion, the Settlement, which provides compensation for dementia, Alzheimer's disease, Parkinson's disease, and ALS, compensates the key alleged impairments associated with CTE based on the current state of the science.

49. The McKee Study is particularly important for understanding this opinion. As stated above, in the McKee Study, the authors found that the symptoms most associated with CTE III and CTE IV were memory problems, attention and learning problems, and executive dysfunction. McKee Study at 55-63. They found that subjects with CTE III and IV essentially had progressed to dementia. *Id.* at 59. They also found that many of the subjects had comorbid, *i.e.*, co-occurring, disease with CTE. Those comorbid diseases were Alzheimer's disease, Parkinson's disease, ALS, and frontotemporal dementia. *Id.* Thus, by compensating for dementia and other conditions that have been identified as comorbid with CTE, the vast majority of retired players who manifest symptoms of CTE while living will be compensated according to the studies relied upon by objectors.

50. Objectors maintain that a vast number of mood and behavioral symptoms, which they claim are part of the diagnostic and clinical profile of CTE, remain

uncompensated in the Settlement. But many of these mood and behavioral symptoms are associated with dementia, and are covered under the cognitive domain, which are tested for in the Settlement—namely, learning and memory impairment, impaired concentration and attention, executive dysfunction, language impairment, and visuospatial difficulties. (*See* Exhibit 2 to Settlement Agreement (Test Battery).) Tellingly, based on the McKee Study and the Stern Study, it appears that almost all players with CTE have some impairment in these cognitive domains.

51. I also note the McKee Study concluded that the vast majority of retired football players in the study had CTE III or IV or CTE plus co-morbid disease. *See* McKee Study at 59. These retired players would have been compensated under the settlement while living based on my review of the injury definitions and test battery.

52. Therefore, even based on the limited information available to us today, the Settlement compensates symptomatic players with CTE by compensating for dementia and other conditions that have been identified as comorbid with CTE.

I declare under penalty of perjury that the foregoing is true and correct.

Dated: Chicago, Illinois

November 12, 2014

Julie Ann Schneider, M.D.

EXHIBIT A

CURRICULUM VITAE

JULIE ANN SCHNEIDER, M.D., M.S.

OFFICE ADDRESS

Rush Alzheimer's Disease Center
Rush University Medical Center
600 S. Paulina St., Suite 1022F AAC
Chicago, Illinois 60612
312-942-2360

LABORATORY ADDRESS

Rush Alzheimer's Disease Center Laboratory
Rush University Medical Center
1735 W. Harrison St., Cohn 436
Chicago, IL 60612
(312) 563-3550

CLINIC ADDRESS

Rush Memory Clinic
Rush University Medical Center
600 S. Paulina, Ste 130
Chicago IL 60612
312-942-3333

CURRENT POSITIONS

Professor of Pathology (Neuropathology), Department of Pathology, Rush University Medical Center

Professor of Neurological Sciences, Department of Neurological Sciences, Rush University Medical Center

Associate Director, Neuropathology Core Leader, Senior Neuropathologist, Rush Alzheimer's disease Center, Rush University Medical Center

BOARDS AND LICENSURE

Licensure - Physician and Surgeon, State of Illinois

Diplomate, American Board of Psychiatry and Neurology, Neurology (1993)

Diplomate, American Board of Pathology, Neuropathology (1995)

Certified, United Council for Neurologic Subspecialties (UCNS), Geriatric Neurology (2011)

EDUCATION

Lane Technical High School; Chicago, Illinois, 1979

University of Illinois; Champaign-Urbana; Biology and Psychology B.S., 1983

University of Illinois, School of Medicine; Chicago, M.D. 1987.,

Rush University Medical Center; Master of Science in Clinical Research (7/2000- 6/2003) (K30 – National Institute of Health sponsored program), M.S. 6/14/2003

HONORS

Phi Beta Kappa, University of Illinois, Champaign-Urbana, 1983

Magna Cum Laude, University of Illinois, Champaign-Urbana, 1983

Alpha Omega Alpha, University of Illinois School of Medicine, Chicago, 1985

PROFESSIONAL EXPERIENCE

Medical Internship, Department of Internal Medicine, Michael Reese Hospital, Chicago IL. July 1987 - June 1988

Neurology Residency, Department of Neurology, University of Chicago, Chicago, IL. July 1988 - June 1991

Neuropathology Fellowship, Department of Pathology (Neuropathology), University of Chicago, Chicago, IL. July 1991 - June 1993

Mount Sinai Hospital and Neurosciences Limited, Chicago Illinois (Part-time clinical neurology), 1991 - 1993

Neuropathology Fellowship, Department of Pathology and Laboratory Medicine (Neuropathology), Emory University School of Medicine, Atlanta GA. July 1993-June 1994

Instructor of Pathology (Neuropathology), Department of Pathology and Laboratory Medicine, Emory University School of Medicine, Atlanta. July 1994-May 1995

Instructor of Neurology (joint appointment), Department of Neurology, Emory University School of Medicine, Atlanta. July 1994-May 1995

Emory Neurobehavioral Program, Wesley Woods Geriatric Hospital. Memory Assessment Clinic (outpatient clinic) Geriatric Neurology/Neuropsychiatry Inpatient Service. July 1994 -July 1996

Assistant Professor of Neuropathology, Department of Pathology & Laboratory Medicine, Emory University School of Medicine, Atlanta. June 1995 -July 1996

Assistant Professor of Neurology (joint appointment), Department of Neurology, Emory University School of Medicine, Atlanta. July 1995-July 1996

Assistant Professor of Neurology, Rush Alzheimer's Disease Center, Department of Neurological Sciences, Rush University Medical Center; August 1996-present.

Assistant Professor of Pathology (Neuropathology) (joint appointment), Department of Pathology, Rush University Medical Center. August 1996- November 2005.

Associate Professor of Pathology (Neuropathology), Department of Pathology, Rush University Medical Center; November 2005 -2012.

Associate Professor of Neurology, Rush Alzheimer's Disease Center, Department of Neurological Sciences, Rush University Medical Center; April 2006 -2012.

Associate Director, Neuropathology Core Leader, Rush Alzheimer's disease Core Center [NIH P30AG10161 (Bennett)] September 2011 – current.

Professor of Pathology (Neuropathology), Department of Pathology, Rush University Medical Center. 2013- current

Professor of Neurological Sciences (joint appointment), Department of Neurological Sciences, Rush University Medical Center. 2013 - current

GRANTS AND FUNDING (CURRENT)

1R01AG042210 (Schneider) 07/1/12- 06/30/17

Epidemiologic Study of TDP-43 Pathology in Aging and Dementia
Major goal is to determine whether age-related TDP-43 pathology represents a separate pathologic process associated with a dementia syndrome with a distinct cognitive phenotype and specific genetic risk factors that are separate from AD.
Role: Principal investigator

R01NS078009 (Buchman) 9/15/12-6/30/17
NINDS
The Clinical Profile of Parkinson's Disease (PD) Pathology
The overall goal is to characterize the clinical profile of PD pathology in older persons without a diagnosis of PD.
Role: co-investigator

R01AG043379 (Buchman) 9/30/12-8/31/17
NIA
Brain and Spinal Cord Microvascular Pathology in Late-Life Motor Impairment
The overall goal is to test the hypothesis that specific microvascular pathologies in the brain and spinal cord contribute to late-life motor impairment.

U01AG016976 (Kukull/Montine) 7/1/12-6/30/13
NIA (Pilot)
Optimization of Neuropathologic Assessment of Alzheimer's Disease
The overall goal is to optimize neuropathologic diagnosis of Alzheimer's disease for uniformity and accurancy across centers
Role: Site Principal Investigator

P30 AG10161 (Bennett) 9/30/91-6/30/16
Rush Alzheimer's Disease Core Center
Major goals to provide core infrastructure support for research regarding aging/dementia/AD.
Role: Associate Director, Core Leader: Neuropathology Core

R01 AG031553 (Morris) 3/15/08 – 2/28/13
Epidemiologic Study of Brain Vitamin E, Diet, and Age-Related Neurologic Diseases;
Major goal is analyze Vitamin E in brain, CSF, serum, and diet and compare to neuropathology and dementia.
Role: Co-Principal Investigator

R01 AG17917 (Bennett) 9/30/01-6/30/13
Epidemiologic Study of Neural Reserve and Neurobiology of Aging;
Major goals are to identify structural bases of reserve and examine mechanisms risk factors lead to age-related functional impairment.
Role: Co-Investigator

R01 AG15819 (Bennett) 7/1/98 – 6/30/13
Risk Factors, Pathology and Clinical Expressions of AD
The major goals of this project are to examine the pathologic mechanisms through which risk factors lead to clinical AD.
Role: Co-investigator

P01 AG14449 (Mufson) 9/1/97 – 3/31/13
Neurobiology of Mild cognitive impairment in the Elderly;

The major goals are to identify neurobiologic substrates of mild cognitive impairment.
Role: Co-investigator (Neuropathologist)

R01AG033678-01 (Boyle) 9/15/2009 - 6/30/14
Epidemiologic study of impaired decision making in preclinical Alzheimer's disease.
The overall goal of the proposed epidemiologic study is to examine the causes and consequences of impaired decision-making in old age.
Role: Neuropathologist

R01AG034374 (Boyle) 8/15/2009 -7/31/14
Characterizing the behavior profile of healthy cognitive aging.
Goal is to use innovative statistical approaches to characterize the profile of healthy cognitive aging defined as age-related cognitive change not accounted for by the presence of common neuropathologies (i.e., Alzheimer's disease, cerebral infarcts, and the Lewy body diseases) or terminal decline.
Role: Neuropathologist.

P01 AG09466 (deToledo-Morrell) 4/1/1991-8/31/12
Anatomic, Physiologic, Cognitive Pathology of AD; major goals- to identify neurobiologic, radiologic, physiologic, cognitive markers with AD;
Role: Co-Investigator; Neuropathologist, Administrative Core

R01 AG36042 (Bennett) 9/15/09-8/31/14
Exploring the Role of the Brain Epigenome: Cognitive Decline and Life Experiences
The goal of the study is to examine the relation of DNA methylation to cognitive decline and life experiences.
Role: Neuropathologist

R01 HL096944 (Levine) 9/1/09 – 6/30/13
Stroke and aPL: Community-Based Clinicopathologic Study
The major goal of this project is to investigate the role and mechanisms of antiphospholipid antibodies (aPL) in the development of pathologically-proven ischemic brain infarction.
Role: Neuropathologist

R01AG040039 (Arvanitakis) 9/30/11-8/31/16
National Institute on Aging
Vascular Cognitive and Motor Decline: Impact of aPL
The major goal of this project is to investigate the role and mechanisms of antiphospholipid antibodies (aPL) in the development of cognitive and motor decline in aging.

R01AG039478 (Arnold) 4/1/11-3/31/16
National Institute on Aging
Targeted Proteomics of Resilient Cognition in Aging

The major goal of the study is to identify candidate proteins and pathways that best confer cognitive resilience despite the accumulation of neurodegenerative disease pathology.
Role: Neuropathologist

R01AG036836 (De Jager) 9/15/11-8/31/15
National Institute on Aging
Exploring the Role of the Brain Transcriptome in Cognitive Decline
The major goal is to investigate the transcriptome of human brain tissue to identify molecular pathways that contribute to cognitive decline.
Role: Neuropathologist

GRANTS AND FUNDING (PAST)

R01 AG030146 (Evans) 9/30/07 – 7/31/12

Genetic Epidemiology of Cognitive Decline in an Aging Population Sample
The major goal is to conduct a genome-wide association scan of 550,000 SNPs to identify loci associated with cognitive decline.
Role: Co-Investigator

R01 AG011101 (Evans) 3/1/93 – 8/31/12

Risk Factors for Incident Alzheimer's Disease in a Biracial Community
The major goal is to identify potentially reversible risk factors for Alzheimer's disease and MCI in a biracial longitudinal population-based study.
Role: Co-Investigator

RC2 AG36547 (Bennett) 9/30/09-8/31/12
Cognitive Decline and Dementia: Life Experiences and the Brain Histone Epigenome
The goal of the study is to examine the relation of histone acetylation to cognitive decline and life experiences.
Role: Neuropathologist

R21 AG030346 (Kelly) 9/30/08 – 8/31/12
Relationship Between AD Clinicopathological Changes and CNS Sex Steroid Hormones;
Major goal is to determine the relationship between brain tissue hormone levels and cognitive impairment, and AD pathology.
Role: Neuropathologist

R01 AG024480 (Buchman) 7/1/05 – 6/30/12
Risk Factors and the Neurobiologic Substrate of Frailty
Major goals of this project are to identify the neurobiologic substrate of physical frailty and identify risk factors for frailty.
Role: Co-Investigator

R01 AG24871 (Wilson) 7/1/06 – 4/30/11
Neurobiologic Study of Psychological Distress & Dementia;
Major goal to identify the mechanisms underlying the association of chronic psychological distress with dementia.
Role: Co-investigator (Neuropathologist)

R21 AG30765 (Bennett) 9/1/07 – 6/30/10
Degraded Rationality: Subclinical Neuropathology and Neuroeconomic Behavior in Older Persons
Role: Co-Investigator

K- 23 AG23675 (Arvanitakis) 01/15/05 – 12/31/09
National Institute on Aging
Oxidative Stress, Aging, and Alzheimer's Disease
Role: Consultant

K23 - AG023040 (Boyle) 5/1/04 - 4/30/09
National Institute on Aging
Role: Mentor
Effects of Vascular Disease in Mild Cognitive Impairment
The major goals are to examine the correlates, predictors, and consequences of mild cognitive impairment with an emphasis on vascular disease.
Role: Consultant

R01 AG021972 (Morris) 8/15/04 – 7/31/09
Long-Term Dietary Risk Factor Assessment and Incident AD;
Major goals of this project are to determine the relationship between dietary Vitamin E patterns from foods and supplements and the subsequent risk of incident Alzheimer's disease.
Role: Co-Investigator

Rush Scientific Leadership Council Grant Awards (Schneider) 7/1/2008
Translational Science Consortium, Capital Equipment Grant Award
Capital Research Equipment (Leica BOND^{TM} Fully integrated IHC and ISH)

K08 AG00849 (Schneider) 3/01/00-2/28/05
Epidemiology, Pathology and Parkinsonism in Aging.
Major goals: Examine the relation of nigral pathology to parkinsonism in aging and AD.
Role: Principal Investigator

P30AG10161 NACC Collaborative Grant (Weintraub) 7/01/05 – 6/30/07
National Alzheimer's Coordinating Centers Collaborative Grants
Clinical Phenotypes of FTD are Determined by Neuropathologic and Biochemical Features

Role: Site principal investigator

P30 AG10161 Rush Alzheimer's disease Pilot Award (Schneider) 7/01/05-6/30/06
Vitamin E, the Aging Brain, and Cognitive and Motor Impairment in Aging
Major goals: Investigate brain Vitamin E and the relationship between Vitamin E and brain pathology and neural reserve.
Role: Principal Investigator

P30AG10161 NACC Collaborative Grant (Kowal) 7/00/00 - 6/30/01
National Alzheimer's Coordinating Centers Collaborative Grants
NACC Vascular pathology Consortium
Role: Site principal investigator

Illinois Department of Public Health; AD Research Funds (Schneider) 7/1/98-6/30/00.
A beta 40 and A beta 42 and memory impairment in aging and Alzheimer's disease.
Role: Principal investigator: FY98-00

P30AG010130 NIA Emory Alzheimer's disease center (Mirra) 7/1/94-6/30/96
Role: Co-investigator: Neuropathology and Clinical Cores

PROFESSIONAL ORGANIZATIONS

McKinley Health and Advisory Board; Voting member, Publicity, Needs Assessment, and Search Committees, 1981 - 1983

Secretary and Treasurer of Alpha Chapter, Alpha Omega Alpha , 1986

American Academy of Neurology (AAN), 1990 – present

Behavioral Neurology Section of the AAN, 1995 - present

Geriatric Neurology Section of AAN, 2005 – present

Ethics Section of AAN, 2006 - present

American Association of Neuropathologists, 1994 – present

American Heart Association, 2014 – present

Board of trustees, International CAA Society, 2014 - present

PROFESSIONAL ACTIVITIES

Manuscript peer review
Alzheimer's disease and Associated Disorders

American Journal of Neuroradiology
American Journal of Pathology
Annals of Neurology
BMC Neurology
Brain
Brain pathology
Current Alzheimer's Research
Dementia and Geriatric Cogn. Dis.
European Journal of Neurology
FASEB Journal
JAMA
Journal of Alzheimer's disease
Journal of Experimental Neurology
Journal of Geriatric Psychiatry and Neurology
Journal of the American Geriatrics Society
Journal of Gerontology
Journal of Histochemistry and Cytochemistry
Journal of Neurology
Journal of Neuropathology and Experimental Neurology
Lancet
Lancet Neurology
Molecular Psychiatry
Movement Disorders
Nature Reviews Neurology
Neurology
Dementia and Geriatric Cognitive Disorders
Stroke

Editorial Boards

2005-	Monitoring editor, Journal Histochemistry and Cytochemistry
2008-	Editorial Board, International Journal of Clinical and Experimental Pathology (IJCEP, ISSN 1936-2625)
2010-2011	Associate editor, Journal of Alzheimer's Disease
2013	Guest editor, Alzheimer Disease & Associated Disorders - An International Journal
2013-	Editorial Board, Journal of Neuropathology and Experimental Neurology.

NIH grant reviews

2006	National Institute on Aging; Training (T32-T35) Grant Review Committee, ad-hoc member

2008	National Institute on Aging; Alzheimer' Disease Research Center (ADRC) Grant Review Committee
2009	National Institute on Aging, P01 Review Committee (Einstein Aging Study, Richard Lipton)
2009	National Institute on Aging; Alzheimer' Disease Research Center (ADCC) Grant Review Committee
2009 -13	Standing member, National Institute on Aging; Neuroscience of Aging Review Committee
2012-2013	Chair, National Institute on Aging; Neuroscience of Aging Review Committee
2014	National Institute on Aging; Alzheimer' Disease Research Center (ADRC and ADCC) Grant Review Committee

Other Grant peer review

1998	Blowitz-Ridgeway Foundation
1999	Loyola School of Medicine Intramural Grants
1999- 2007	Alzheimer's Association Grant review
2001	The Retirement Research Foundation Grants
2001	Basic Life Sciences UIC Campus Research Board (CRB
1999- 2008	Alzheimer's Disease Research Fund Review Panel, Illinois Department of Public Health
2014	Wellcome Trust Grant review, ad hoc

Abstract peer review

2008-2010	Aging and Dementia abstracts; American Academy of Neurology Annual Meetings.
2011,2013	VASCOG Society: The International Society of Vascular Behavioural and Cognitive Disorders.
2012-2014	American Association of Neuropathologists Meeting

2013	Alzheimer's Association International Conference (AAIC), Boston July 13-18
2014	Alzheimer's Imaging Consortium (AIC) preconference to AAIC, Copenhagen

Elected committees:

2001- 2002	Awards committee, American Association of Neuropathologists
2011-	Program Committee, American Association of Neuropathologists
2011-	Neuropathology Core Steering Committee; National Alzheimer's Disease Centers

Rush University Medical Center:

1996-2009	Neuropathology Quality Assurance Committee
1997	Committee for Rush Medical Student applicant interviews
1997- present	Rush Alzheimer's Disease Center Tissue Distribution Committee
1997- 2005	Woman's Health Initiative – Memory Study; Examining Neurologist for Rush site
2000- present	Rush Alzheimer's Disease Center Executive Committee
7/2005 – 12/2008	Department of Neurological Sciences Advisory Committee
5/2007 - present	Rush Biospecimen Storage Committee
9/2011- present	Neuropathologist, Rush Hospital and Medical Center Brain Autopsy Service
1/2011- present	Rush Scientific Leadership Council and Pilot Grant Committee

Other Academic Activities

7/1/2006	Invited Member, Alzheimer's Disease Center *Biospecimen Task Force*, National Institute on Aging
8/2007	Invited participant, *NIA Genetics of Alzheimer's Disease Planning Meeting,* Bethesda, National Institute on Aging

12/2007	Consultant, Neuropathology subcommittee for the AD Genetics Consortium (ADGC)
4/2008	Invited auditor, American Academy of Neurology Annual Meeting;Program 5PS.005: Poster Session 5; Poster Grouping: Aging and Dementia: Pathology 91 – 100
4/2008	Co-chair, American Academy of Neurology Annual Meeting; Session S11: Aging and Dementia: Basic Science/Neuropathology
10/2008-	Neuropathology consultant, AVID radiopharmaceuticals
2009 -2012	Neuropathologist, Bielschowsky Histology Core laboratory for Avid AV-45-A07, A16 Phase 3 Clinical Trial - Autopsy Protocol Study.
11/2008- 12	Geriatric Neurology Examination Committee – United Council for Neurologic Subspecialties
3/2009	Co-chair; 7th Annual Mild Cognitive Impairment (MCI) Symposium Sponsored by the Behrman Center for Medical Education at Mount Sinai Medical Center, Miami FL; Session; LBD and Vascular Cognitive Impairment.
10/2009	AHA writing committee initiated by the Stroke and EPI Councils on the topic of vascular cognitive impairment (officially titled "Vascular Contributions to Cognitive Impairment and Dementia")
8/2009	Invited Reviewer; Continuum: Lifelong Learning in Neurology, a program of the American Academy of Neurology; Special issue Dementia.
3/2010	Invited Speaker and Participant, Vascular mechanisms in Brain Aging UK-USA (LLHW-NIA) Centres Workshop.
4/2010	Co-chair, American Academy of Neurology Annual Meeting; Session S. Toronto, Canada
5/2010	Consultant, Advisory Board on "Alzheimer's diagnosis and the role of Amyloid Imaging", GE Healthcare
9/2011	Steering Committee, National Institute of Aging/ Alzheimer's Association sponsored revision of the NIA-Reagan criteria for a pathologic diagnosis of Alzheimer's disease.

6/2011	Scientific Committee; VASCOG conference 2011 Lille France;
7/2012	Chair; Alzheimer's Association International Conference (AAIC) session entitled Related Dementias 2: Frontotemporal Dementia and Dementia with Lewy Bodies. Vancouver, British Columbia, Canada.
7/2013	Invited, Scientific Committee; VASCOG conference 2013 Toronto, Canada
6/2014	Consultant, Navidea Biopharmaceuticals Inc.
10/2014	Alzheimer's expert panel, CME video expert panel; "Advances in Alzheimer's Disease: Early Imaging and Therapeutics", sponsored by Eli Lily Corporation.

EXTERNAL ADVISORY BOARDS

11/2004 - 2013	External Advisor for P01 AG12435; National Institute on Aging; *Aging Brain: Vasculature, Ischemia*, and Behavior – Principal Investigator: Dr. Helena Chui 9/30/04 –
10/2008 - present	Scientific External Advisory Board (U01) National Institute on Aging; *University of Washington/Group Health Alzheimer's Disease Patient Registry/Adult Changes in Thought study.*– Principal Investigator: Dr. Eric Larson
3/2011-present	External Advisor Committee, University of California San Francisco Neurodegenerative Brain Bank (NDBB)
11/2011-present	External Advisory Board, Committee, New York University – Alzheimer's Disease Core Center
1/2012-present	External Advisory Board, Boston University – Alzheimer's Disease Core Center.
1/2013- present	Cell Bank Advisory Committee, National Cell Repository for Alzheimer's Disease (PI: Tatiana Foroud).

ADDITIONAL TEACHING EXPERIENCE

Lecturer and Laboratory Assistant, Clinical Pathophysiology Course (Neuropathology), second year medical student lectures, University of Chicago, 1993
Laboratory Teaching Assistant, Neurobiology Course (Neuroanatomy), first year medical student course, University of Chicago, 1993

Neuropathology and clinical conferences, for neurology and pathology residents and faculty, University of Chicago, 1991-1993

Neuropathology course for neurology, neurosurgery, and pathology residents, University of Chicago, 1992-1993

Neuropathologic correlation at weekly Neurology/ Neurosurgery/ Neuroradiology, and Pediatric conferences, Emory University School of Medicine and Egleston Children's Hospital.
1993 -1996

Faculty Lecturer, Neuropathology Section, Pathology 615 - second year medical school course, Emory University School of Medicine, 1994-1996

Brain cutting conference, weekly conference /microscopic sign out with residents, Emory University School of Medicine, 1994 -1996

Tutor, problem based learning, sophomore medical students, Emory University School of Medicine, 1996

Faculty Presenter, *Role of Interdisciplinary Teams in working with patients and families with dementia,* Geriatric Interdisciplinary Team Training Program, 2000

Faculty Lecturer, Medical Student Pathology Laboratory, Brain Pathology; Rush University Medical Center; 2004 – 2009

Faculty Lecturer, Neuropathology course for Neurosurgery residents at Rush University Medical Center, 2001-present.

Faculty Lecturer, Neuropathology course for Neurology residents at Rush University Medical Center, 2009-present.

THESIS COMMITTEES

Member, Thesis Committee (Brinda Desai, PhD candidate, Rush University Neuroscience Graduate Program). Thesis successfully defended 1/2009

Member, Thesis Committee (Aditi Patel, PhD candidate, Rush University Neuroscience Graduate Program. Thesis successfully defended 7/2011

INVITED PRESENTATIONS (RUSH UNIVERSITY MEDICAL CENTER)

Speaker, *Neuropathology of Dementia*, Pfizer Educational Conferences, Rush University Medical Center and Rush Alzheimer's Disease Center, 1997.

Speaker, *Neuropathology of Dementia*, Bayer Educational Conferences, Rush University Medical Center and Rush Alzheimer's Disease Center, 1998.

Speaker, *Treatment of Alzheimer's disease,* sponsored by Pfizer Inc.; Rush University Medical Center Internal Medicine; Round table discussion. Chicago IL 1998

Speaker, *Alzheimer's disease and related disorders*, Leadership of Dementia Special Care Units Certificate Course, , Rush University Medical Center and Rush Alzheimer's Disease Center, 1999.

Speaker, *NonAlzheimer's Dementias Workshop:* Neurology for the Non-Neurologists Annual Conference, sponsored by Dept of Neurological Sciences, Rush University Medical Center 2000-2001

Speaker, *The Neuropathology of Alzheimer's disease,* Preparing Leaders for the Future of Dementia Care: Dementia Special Care Unit Director Certificate Course at Rush University Medical Center. 2002-2004

Speaker, *Parkinsonian Signs in Older Persons, Epidemiology and Pathology*, Grand Rounds, Department of Neurological Sciences, Rush University Medical Center 2003.

Speaker, *The Neuropathology of Alzheimer's disease,* Primary Provider Group at Rush-Presbyterian-St. Luke's Medical Center. Sponsored by the Rush Alzheimer's Disease Center. 2001 – 2004

Speaker, *Apolipoprotein E, Alzheimer's Disease Pathology, and Cerebral Infarctions*, Grand Rounds, Department of Neurological Sciences, Rush University Medical Center 2004.

Speaker, *The Neuropathology of Dementia*; Annual Unit Director's Course, Rush University Medical Center 5/2005

Speaker, *The Neuropathology of Cognitive Impairment and Dementia, an Update from the Religious Orders Study.* Grand Rounds, Department of Neurological Sciences. Rush University Medical Center 1/2006

Speaker, *The Neuropathology of Parkinsonism in older Persons (data from K08 AG00849)* . Research on Aging Conference, Rush University Medical Center and Rush Alzheimer's Disease Center. 1/06

Speaker, *The Neuropathology of Dementia*; Annual Unit Director's Course, Rush University Medical Center 5/2006

Speaker, *Frontotemporal and other atypical dementias-Part II-the neuropathologic perspective* Grand Rounds, Department of Neurological Sciences. Rush University Medical Center 8/2006

Speaker, *The aging brain and risk factors for dementia,* ASSIST meeting for Rush Alzheimer's disease Center, Rush University Medical Center, Chicago IL 3-2008

Speaker, *The Neuropathology of Dementia*; Annual Unit Director's Course, Rush University Medical Center 5/2008

Speaker, *Mixed Pathology in Probable Alzheimer's disease and Mild Cognitive Impairment* Grand Rounds, Department of Neurological Sciences, Rush University Medical Center 5/2008.

Speaker, *Alzheimer's disease; Where we are and Where are we going,* Annual CNA conference for Rush Alzheimer's disease center, Rush University Medical Center, Chicago IL March 2008

Speaker, *The Neuropathology of Dementia*; Annual Unit Director's Course, Rush University Medical Center 5/2009

Speaker, *Genetics of Alzheimer's Disease;* Without Warning Annual Conference, Rush Alzheimer's disease center and Rush University Medical Center 10-24-2009

Speaker, Rush Rounds Lecture Series; *Alzheimer's disease*; hosted by Rush Philanthropy and President; 10-05-09

Speaker (Keynote speaker), *Brain autopsy*, Inside Highlights, Rush Alzheimer's disease Center, Rush University Medical Center. December 2009

Speaker, An epidemiologic study of TDP-43 in aging. Neuroepidemiology conference. Rush Alzheimer's disease Center. 09-20-2012

Speaker, "Special Topics Course on Aging"; Department of Immunology/Microbiology & Medicine 4/2014

INVITED PRESENTATIONS (OUTSIDE RUSH)

Speaker, *Brain Tumors*, Psychiatry Board Review Course, Chicago IL 1993

Speaker, *Creutzfeldt-Jakob Disease*, Eye Bank of America, Atlanta GA, 1995

Speaker, *What is new in Alzheimer's disease?* Jewish Family Services, Milwaukee, WI, 1996

Speaker, *Overview of Alzheimer's disease and related dementias, Pharmacologic Management of Behavioral disorders in Alzheimer's disease, The Religious Orders Study and Aging in the 21st century,* Educational Conferences for Members and Staff of Communities participating in the

Religious Study, and Educational Courses for Family Caregivers and Professionals, Dubuque Iowa and St. Cloud MN, 1997 – 1999

Speaker, *Current Trends in Dementia Research and Care*, 5 session course for Family Caregivers and Professionals, Condell Day Center, IL 1999

Speaker (Guest Faculty Lecturer), *Alzheimer's disease,* Medical Neuroscience course, Finch University of Health Sciences/The Chicago Medical School, 1999

Speaker, *Dementia and Alzheimer's disease: A review and update.* Illinois Masonic Department of Internal Medicine Grand Rounds 1999.

Speaker, *Applying post-mortem neurobiologic indices in epidemiologic studies of Alzheimer's disease.* Buck Center, Research in Aging. San Francisco CA. 2000

Speaker, *Update on Alzheimer's Disease:* Midwest Clinical Conference 2001 – Strategic Healthcare for a Diverse Population. Chicago Medical Society. Navy Pier – Chicago.

Speaker, *Risk factors for dementia - How neuropathology can provide clues in epidemiologic studies*, Cellular and Behavioral Mechanisms of Aging and Dementia, Northwestern University Chicago IL 3/2005

Speaker, *The Aging Brain and Risk Factors for Cognitive Impairment in Older Persons*, 2nd Annual Regional CME meeting: Providing Quality Dementia Care: The Critical Role of the Primary Care Clinician; Washington University School of Medicine, St. Louis. 6/2005

Invited Faculty Course Speaker, *Vascular Dementia and Dementia with Lewy bodies*, in 7BS.005 Dementia Evaluation in the Office, Breakfast Seminar for American Academy of Neurology Educational Conference, Annual Meeting 2006-2008. 4/15/2005-7 .

Speaker, *Co-existence of Vascular and Alzheimer Pathology in Cognitively Impaired Individuals.* 4th Annual Mild Cognitive Impairment (MCI) Symposium. Sponsored by the Behrman Center for Medical Education at Mount Sinai Medical Center, Miami FL. Feb 24-26, 2006.

Speaker (Keynote Speaker), *The Aging Brain and Risk Factors for Cognitive Impairment in Older Persons*, 11th Annual Conference on Alzheimer Disease and Related Disorders, Springfield, Illinois (Sponsored by Southern Illinois University School of Medicine, Center for Alzheimer Disease and Related Disorders; Illinois Department on Aging and the Alzheimer's Association). May 25, 2006

Symposium Speaker, *Mixed Pathologies In Alzheimer's Disease And Mild Cognitive Impairment*, International Conference on Alzheimer's Disease and Related Disorders. Chicago IL 7/2008

Speaker (Keynote), Update on Research – *Alzheimer's disease and Frontotemporal dementias; Annual Research Update*. Alzheimer's Association, St. Louis Chapter November 5th, 2008

Speaker, The *neuropathology of MCI and probable Alzheimer's disease* 7th Annual Mild Cognitive Impairment (MCI) Symposium. Sponsored by the Behrman Center for Medical Education at Mount Sinai Medical Center, Miami FL. March 27-28, 2009.

Speaker, *Biological metals, fatty acids, and cognitive function in older persons,* Biological Applications of X-Ray Microprobes, sponsored by Argonne National Laboratory, National Center for Research Resources, BioCAT. Prentice Women's Hospital, Chicago, IL. Nov 15-16th, 2007.

Speaker, ARCS Foundation, Chicago Chapter; *Cognitive and Motor Changes with Aging* 11-09

Speaker, *Vascular mechanisms in Brain Aging*. UK-USA (LLHW-NIA) Centres Workshop. Atlanta Georgia; March 15-16, 2010

Invited Faculty Course Speaker, *Neuropathological correlates of normal aging and MCI*, in 1PC.002. Is it normal aging or MCI? Half-day course; American Academy of Neurology; Annual Meeting, Hawaii 2011.

Speaker, (Keynote); Where Alzheimer's disease meets vascular disease; Second International Symposium of the Alzheimer & Dementia Center at the Methodist Neurological Institute in Houston, Texas, March-6-2012;

Speaker, "What Every Neuropathologist Needs to Know: NIA-AA Revised Guidelines for the Diagnosis of Alzheimer's Disease. Eighty-Eighth Annual Meeting of the American Association of Neuropathologists. Chicago IL, June 23, 2012.

Speaker, "Microvascular disease and Cognition" 3rd International CAA Conference. Cerebral Amyloid Angiopathy and Related Microangiopathies. Leiden University Medical Center, Leiden, The Netherlands. October 2012.

Speaker, "Epidemiology of Vascular Contributions to Alzheimer's disease & Dementia"; Vascular Contributions to Alzheimer's & Dementia meeting. Alzheimer's Association developed in collaboration with the National Institute of Neurological Disorders, Chicago; December 17, 2013.

Speaker, Vascular Dementias Committee, AD-Related Dementias Workshop, National Institute of Aging, May 1-2 2013

Speaker, "The impact of small vessel disease on Cognition in patients with Alzheimer's disease". International Stroke Conference 2/2014, San Diego CA.

Speaker, Cognitive Impairment In Aging: What We Have Learned From Longitudinal Clinical-Pathologic Studies., 9th BARCELONA-PITTSBURGH Conference Program 4/23/2014

Speaker, "Hippocampal Sclerosis of Aging". Alzheimer's Association International Conference. Copenhagen, Denmark. 7/2014

Speaker, "Update on National Alzheimer's Centers Coordinating Center Neuropathologic data collection guidelines". Alzheimer's disease Neuropathology Core Meeting, Baltimore 10/21/14.

Speaker, "Overlapping Neuropathologies in AD: Impact on Drug Development", Alzheimer's Association Research Roundtable. 10/2014

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BOOK CHAPTERS

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